Refine Search

Search Results -

Terms	Documents
L13 and adenovirus adj vector	0

Database: US Pre-Grant Publication Full-Text Database; US Patents Full-Text Database; US OCR Full-Text Database; EPO Abstracts Database; JPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L20

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Search History

DATE: Wednesday, February 15, 2006 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=EPAB; THES=ASSIGNEE; PLUR=YES; OP=ADJ			
L20	L13 and adenovirus adj vector	0	L20
DB=DWPI; THES=ASSIGNEE; PLUR=YES; OP=ADJ			
L19	L13 and adenovirus adj vector	0	L19
DB=USPT; THES=ASSIGNEE; PLUR=YES; OP=ADJ			
L18	L13 and adenovirus adj vector	10	L18
L17	L16 and adenovirus	45	L17
L16	L15 and adeno	45	L16
L15	L13	127	L15
DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; THES=ASSIGNEE; PLUR=YES; OP=ADJ			
L14	L13 and L3	0	L14
L13	L12 and Hela	300	L13
L12	L10 and host adj cell	1048	L12
L11	L10 and L3	0	L11
L10	S adj 3	76043	L10
L9	(WO009911764)	0	L9
L8	L4 and (WO009911764)	0	L8

L7	L4 and (WO009911764)	0	L7
DB=USPT; THES=ASSIGNEE; PLUR=YES; OP=ADJ			
L6	L5 and (WO009911764)	0	L6
L5	L4	83	L5
DB=PGPB,USPT,EPAB,JPAB,DWPI; THES=ASSIGNEE; PLUR=YES; OP=ADJ			
L4	L3 and adenovirus	88	L4
L3	435/367	159	L3
L2	(435/367.ccls and adenovirus)![IPC]	0	L2
L1	(435/367)![IPC]	0	L1

END OF SEARCH HISTORY

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 NEWS  5  DEC 14  2006 MeSH terms loaded for MEDLINE file segment of TOXCENTER
 NEWS  6  DEC 14  CA/CAplus to be enhanced with updated IPC codes
 NEWS  7  DEC 21  IPC search and display fields enhanced in CA/CAplus with the
                  IPC reform
 NEWS  8  DEC 23  New IPC8 SEARCH, DISPLAY, and SELECT fields in USPATFULL/
                  USPAT2
 NEWS  9  JAN 13  IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
 NEWS 10  JAN 13  New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to
                  INPADOC
 NEWS 11  JAN 17  Pre-1988 INPI data added to MARPAT
 NEWS 12  JAN 17  IPC 8 in the WPI family of databases including WPIFV
 NEWS 13  JAN 30  Saved answer limit increased
 NEWS 14  JAN 31  Monthly current-awareness alert (SDI) frequency
                  added to TULSA

 NEWS EXPRESS  JANUARY 03 CURRENT VERSION FOR WINDOWS IS V8.01,
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               V8.0 USERS CAN OBTAIN THE UPGRADE TO V8.01 AT
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FILE 'BIOSIS' ENTERED AT 08:54:53 ON 15 FEB 2006

=> "CCL-2.2"
L1 5 "CCL-2.2"

=> adenovirus
L2 57003 ADENOVIRUS

=> "hela s3"
L3 2713 "HELA S3"

=> L2 and L3
L4 24 L2 AND L3

=> L2 and L1
L5 0 L2 AND L1

=> vector and L4
L6 3 VECTOR AND L4

=> D L6 IBIB ABS 1-3

L6 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:899614 CAPLUS
TITLE: Method for production of oncolytic **adenoviruses**
INVENTOR(S): Kadan, Michael; Kaptur, Ronald; Brousseau, David; Mittelstaedt, Denise; Li, Yuanhao
PATENT ASSIGNEE(S): Novartis Ag, Switz.
SOURCE: PCT Int. Appl.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004092348	A2	20041028	WO 2004-US11855	20040415
WO 2004092348	A3	20050310		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2005095705	A1	20050505	US 2004-824796	20040414
PRIORITY APPLN. INFO.:			US 2003-463143P P	20030415

AB **HeLa-S3** cells comprising replication-competent **adenovirus vectors** are provided. Also provided are **HeLa-S3** producer cell lines and methods for producing replication-competent **adenovirus** using the same.

L6 ANSWER 2 OF 3 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1999:111560 BIOSIS
DOCUMENT NUMBER: PREV199900111560
TITLE: Expression of coxsackie-**adenovirus** receptor (CAR) and integrins in a cell line derived from anaplastic large cell lymphoma (ALCL).
AUTHOR(S): Turturro, F.; Link, C. J.
CORPORATE SOURCE: Human Gene Therapy Res. Inst. John Stoddard Cancer Cent., Iowa Methodist Med. Cent., Des Moines, IA, USA

SOURCE: Blood, (Nov. 15, 1998) Vol. 92, No. 10 SUPPL. 1 PART 1-2, pp. 381B. print.
Meeting Info.: 40th Annual Meeting of the American Society of Hematology. Miami Beach, Florida, USA. December 4-8, 1998. The American Society of Heamatology.
CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Mar 1999
Last Updated on STN: 12 Mar 1999

L6 ANSWER 3 OF 3 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1997:45643 BIOSIS
DOCUMENT NUMBER: PREV199799344846
TITLE: Method for targeted gene transfer to liver using DNA-protein complexes.
AUTHOR(S): Findeis, Mark A. [Reprint author]; Wu, Catherine H.; Wu, George Y.
CORPORATE SOURCE: Pharm. Peptides Inc., Cambridge, MA, USA
SOURCE: Robbins, P. D. [Editor]. (1997) pp. 135-152. Methods in Molecular Medicine; Gene therapy protocols.
Publisher: Humana Press Inc., Suite 808, 999 Riverview Drive, Totowa, New Jersey 07512, USA.
ISBN: 0-89603-307-4 (paper), 0-89603-484-4 (cloth).
DOCUMENT TYPE: Book; (Book Chapter)
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Feb 1997
Last Updated on STN: 4 Feb 1997

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FILE 'CAPLUS, BIOSIS' ENTERED AT 08:54:53 ON 15 FEB 2006
L1 5 "CCL-2.2"
L2 57003 ADENOVIRUS
L3 2713 "HELA S3"
L4 24 L2 AND L3
L5 0 L2 AND L1
L6 3 VECTOR AND L4

FILE 'STNGUIDE' ENTERED AT 08:57:05 ON 15 FEB 2006

=> D L4 IBIB ABS 1-24
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L4 ANSWER 1 OF 24 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:244695 CAPLUS

DOCUMENT NUMBER: 143:58180
TITLE: Safety characterization of HeLa-based cell substrates used in the manufacture of a recombinant adeno-associated virus-HIV vaccine
AUTHOR(S): Tatalick, Lauren M.; Gerard, Christopher J.; Takeya, Ryan; Price, David N.; Thorne, Barbara A.; Wyatt, Lisa M.; Anklesaria, Pervin
CORPORATE SOURCE: Targeted Genetics Corporation, Seattle, WA, 98101, USA
SOURCE: Vaccine (2005), 23(20), 2628-2638
CODEN: VACCDE; ISSN: 0264-410X
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The use of transformed cell substrates for prophylactic vaccine manufacturing is widely debated. Extensive characterization is required to address the suitability of neoplastic cell substrates for vaccine manufacture The HeLa-based cell substrate used in the manufacture of a prophylactic rAAV-HIV vaccine, AAV2-gagPRΔRT (tgAAC09) was tested in vivo for its tumor-forming potential, the oncogenic potential of its high mol. weight DNA and the potential presence of occult oncogenic adventitious agents. This data from these in vivo studies, in conjunction with prion gene and protein characterization, cell and viral clearance studies and quantity of residual host-cell DNA levels in the purified tgAAC09 vaccine, were used to establish what the authors believe to be an acceptable safety profile for the vaccine manufacturing process. The tumor-producing dose in 50% of the animals was consistent with that in a published report from FDA staff for HeLa cells. High mol. weight cellular DNA was not oncogenic and no occult oncogenic agents were detected by testing in nude mice and newborn rodent models, resp. Endogenous prion protein was also normal and genomic sequence anal. detected no mutations associated with increased risk of prion disease. In addition, the purification process used to produce this vaccine candidate removed all detectable cells (clearance of greater than 22 log10), viral clearance study showed 6-17 log10 clearance of three model viruses and host-cell DNA in the bulk product was less than 100 pg host-cell DNA per dose of 3+1011 DNase resistant particles (DRP) of the vaccine. Taken together, the data from the in vivo and in vitro tests that were performed to characterize the HeLa based producer cell line (T3B12-5B) and **HeLa S3** cells support the use of these cells as substrates for the manufacture of a purified rAAV-HIV vaccine candidate. The data also supports the ability of the process, employing the HeLa cell substrate, used to manufacture the rAAV-HIV vaccine to produce a product as free of adventitious agents as current testing procedures can document. Safety of the rAAV-HIV vaccine is currently being assessed in a Phase I clin. trial.

REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 2 OF 24 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:60754 CAPLUS
Correction of: 2004:1036571
DOCUMENT NUMBER: 142:233342
Correction of: 142:16836
TITLE: Sequences of human schizophrenia related genes and use for diagnosis, prognosis and therapy
INVENTOR(S): Liew, Choong-Chin
PATENT ASSIGNEE(S): Chondrogene Limited, Can.
SOURCE: U.S. Pat. Appl. Publ., 156 pp., Cont.-in-part of U.S. Ser. No. 802,875.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 47
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004241727	A1	20041202	US 2004-812731	20040330
US 2004014059	A1	20040122	US 2002-268730	20021009
US 2005191637	A1	20050901	US 2004-803737	20040318

US 2005196762	A1	20050908	US 2004-803759		20040318
US 2005196763	A1	20050908	US 2004-803857		20040318
US 2005196764	A1	20050908	US 2004-803858		20040318
US 2005208505	A1	20050922	US 2004-803648		20040318
US 2004241727	A1	20041202	US 2004-812731		20040330
US 2004241727	A1	20041202	US 2004-812731		20040330
US 2004265869	A1	20041230	US 2004-812716		20040330
US 2005208519	A1	20050922	US 2004-989191		20041115
PRIORITY APPLN. INFO.:			US 1999-115125P	P	19990106
			US 2000-477148	B1	20000104
			US 2002-268730	A2	20021009
			US 2003-601518	A2	20030620
			US 2004-802875	A2	20040312
			US 2004-812731	A	20040330
			WO 2004-US20836	A2	20040621

AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing and monitoring diseases using gene-specific and/or tissue-specific primers. The present invention also describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular treatment regimen. [This abstract record is one of 3 records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

L4 ANSWER 3 OF 24 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:899614 CAPLUS
TITLE: Method for production of oncolytic **adenoviruses**
INVENTOR(S): Kadan, Michael; Kaptur, Ronald; Brousseau, David; Mittelstaedt, Denise; Li, Yuanhao
PATENT ASSIGNEE(S): Novartis Ag, Switz.
SOURCE: PCT Int. Appl.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004092348	A2	20041028	WO 2004-US11855	20040415
WO 2004092348	A3	20050310		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2005095705	A1	20050505	US 2004-824796	20040414
PRIORITY APPLN. INFO.:			US 2003-463143P	P 20030415

AB **HeLa-S3** cells comprising replication-competent **adenovirus** vectors are provided. Also provided are **HeLa -S3** producer cell lines and methods for producing replication-competent **adenovirus** using the same.

L4 ANSWER 4 OF 24 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1996:554297 CAPLUS
DOCUMENT NUMBER: 125:187476
TITLE: Selection of the best target site for ribozyme-mediated cleavage within a fusion gene for **adenovirus** E1A-associated 300 kDa protein

(p300) and luciferase
AUTHOR(S): Kawasaki, Hiroaki; Ohkawa, Jun; Tanishige, Norie; Yoshinari, Koichi; Murata, Takehide; Yokoyama, Kazunari K.; Taira, Kazunari
CORPORATE SOURCE: National Inst. Biosci. Human Technology, Agency Industrial Sci. Technology, Tsukuba Science City, 305, Japan
SOURCE: Nucleic Acids Research (1996), 24(15), 3010-3016
CODEN: NARHAD; ISSN: 0305-1048
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The cellular 300-kDa protein known as p300 is a target for the adenoviral E1A oncoprotein and it is thought to participate in prevention of the G0/G1 transition during the cell cycle, in activation of certain enhancers and in the stimulation of differentiation pathways. To determine the exact function of p300, as a first step we constructed a simple assay system for the selection of a potential target site of a hammerhead ribozyme in vivo. For the detection of ribozyme-mediated cleavage, we used a fusion gene (p300-luc) that considered of the sequence encoding the N-terminal region of p300 and the gene for luciferase, as the reporter gene. We were also interested in the correlation of the GUX rule, for the triplet adjacent to the cleavage site, with ribozyme activity in vivo. Therefore, we selected 5 target sites that all included GUX. The rank order of activities in vitro indeed followed the GUX rule; with respect to the kcat, a C residue as the third base (X) was the best, next came an A residue and a U residue was the worst (GUC > GUA > GUU). However, in vivo the tRNAVal promoter-driven ribozyme, targeted to a GUA located upstream of the initiation codon, had the highest inhibitory effect (96%) in **HeLa S3** cells when the molar ratio of the DNA template for the target p300 RNA to that for the ribozyme was 1:4. Since the rank order of activities in vivo did not conform to the GUX rule, it is unlikely that the rate limiting step for cleavage of the p300-luc mRNA was the chemical step. This kind of ribozyme expression system should be extremely useful for elucidation of the function of p300 in vivo.

L4 ANSWER 5 OF 24 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1994:676563 CAPLUS
DOCUMENT NUMBER: 121:276563
TITLE: Vitronectin receptor antibodies inhibit infection of HeLa and A549 cells by **adenovirus** type 12 but not by **adenovirus** type 2
AUTHOR(S): Bai, Mei; Campisi, Lauren; Freimuth, Paul
CORPORATE SOURCE: Biology Department, Brookhaven National Laboratory, Upton, NY, 11973, USA
SOURCE: Journal of Virology (1994), 68(9), 5925-32
CODEN: JOVIAM; ISSN: 0022-538X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The penton base gene from **adenovirus** type 12 (Ad12) was sequenced and encodes a 497-residue polypeptide, 74 residues shorter than the penton base from Ad2. The Ad2 and Ad12 proteins are highly conserved at the amino- and carboxy-terminal ends but diverge radically in the central region, where 63 residues are missing from the Ad12 sequence. Conserved within this variable region is the sequence Arg-Gly-Asp (RGD), which, in the Ad2 penton base, binds to integrins in the target cell membrane, enhancing the rate or the efficiency of infection. The Ad12 penton base was expressed in Escherichia coli, and the purified refolded protein assembled in vitro with Ad2 fibers. In contrast to the Ad2 penton base, the Ad12 protein failed to cause the rounding of adherent cells or to promote attachment of **HeLa S3** suspension cells; however, A549 cells did attach to surfaces coated with either protein and pretreatment of the cells with an integrin αvβ5 monoclonal antibody reduced attachment to background levels. Treatment of HeLa and A549 cells with integrin αvβ3 or αvβ5 monoclonal antibodies or with an RGD-containing fragment of the Ad2 penton base protein inhibited infection by Ad12 but had no effect on and in some cases enhanced infection by Ad2. Purified Ad2 fiber protein reduced the binding of radiolabeled Ad2 and Ad12 virions to HeLa and A549 cells nearly to

background levels, but the concns. of fiber that strongly inhibited infection by Ad2 only weakly inhibited Ad12 infection. These data suggest that αv-containing integrins alone may be sufficient to support infection by Ad12 and that this pathway is not efficiently used by Ad2.

L4 ANSWER 6 OF 24 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1994:316909 CAPLUS
DOCUMENT NUMBER: 120:316909
TITLE: Incorporation of **adenovirus** into a ligand-based DNA carrier system results in retention of original receptor specificity and enhances targeted gene expression
AUTHOR(S): Wu, George Y.; Zhan, Peili; Sze, Lillian L.; Rosenberg, Arielle R.; Wu, Catherine H.
CORPORATE SOURCE: Sch. Med., Univ. Connecticut, Farmington, CT, 06030, USA
SOURCE: Journal of Biological Chemistry (1994), 269(15), 11542-6
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Adenovirus** type 5 was modified by coupling an asialoglycoprotein-polylysine conjugate to the virus by reactions that activate carbohydrate residues. Wild-type virus modified in this manner had greatly decreased infectivity toward normally susceptible **HeLa S3** (asialoglycoprotein receptor (-)) and SK Hep1 (asialoglycoprotein receptor (-)) cells leaving 91 and 86% viable, resp., after 48 h. However, with Huh 7 (asialoglycoprotein receptor (+)) cells, modified virus retained its infectivity leaving only 19% of cells viable under identical conditions. Modified virus was complexed to DNA in the form of a plasmid, pSVHBV surf, containing the gene for hepatitis B surface antigen as a marker of gene expression. Huh 7, receptor (+), cells treated with modified wild type, and modified replication-defective dL312 virus complexed to DNA raised antigen levels by approx. 13- and 30-fold, resp., compared with asialoglycoproteinpolylysine DNA complex alone. Competition with a large excess of an asialoglycoprotein blocked the enhancement by more than 95%. Using a β-galactosidase marker gene, the number of cells transfected by modified virus was found to be 200-fold higher than complex alone. Yet, specificity was retained exclusively for asialoglycoprotein receptor-bearing cells. These data indicate that **adenovirus** can be chemical modified by coupling ligands resulting in targeted gene expression dictated specifically by receptor recognition of the attached ligand.

L4 ANSWER 7 OF 24 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1985:466423 CAPLUS
DOCUMENT NUMBER: 103:66423
TITLE: Analysis of N-methyl-N'-nitro-N-nitrosoguanidine (MNNG)-induced DNA damage in tumor cell strains from Japanese patients and demonstration of MNNG hypersensitivity of Mer- xenografts in athymic nude mice
AUTHOR(S): Watatani, Masahiro; Ikenaga, Mituo; Hatanaka, Toshihiro; Kinuta, Masakatsu; Takai, Shinichiro; Mori, Takesada; Kondo, Sohei
CORPORATE SOURCE: Sch. Med., Osaka Univ., Osaka, 553, Japan
SOURCE: Carcinogenesis (1985), 6(4), 549-53
CODEN: CRNGDP; ISSN: 0143-3334
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Among 15 human tumor cell strains from Japanese patients, 1 strain derived from a patient with thyroid cancer showed the inability to support the growth of **adenovirus** 5 treated with MNNG [70-25-7]. When plated on this Mer- strain, **adenovirus** 5 showed 3-4 times higher sensitivity to MNNG-induced killing than when plated on any of the other 14 Mer+ tumor cell strains. Biochem. anal. showed that the Mer- strain was defective in demethylation repair of O6-methylguanine [20535-83-5] produced by MNNG treatment. The sensitivities of 12 of 15 human tumor strains, including the Mer- strain, to MNNG were compared by measuring

their colony-forming abilities. All the strains tested showed the Rem- phenotype (having higher sensitivity to MNNG-produced cell killing than normal fibroblasts). The differential killing effects of MNNG on Mer- and Mer+ tumor cells under in vivo conditions were tested using the Mer+ **HeLa S3** strain and its Mer- variant. Mer+ and Mer- cells were implanted s.c. into the left and right flanks, resp., of 10 nude mice and the next day, MNNG solution (0.25 mL at 1 mg/mL) was injected into the implantation site of 8 mice. Mer- tumor cells in 6 of 8 treated mice showed no growth and those in the other 2 mice did grow, but regressed after .apprx.3 wk. In contrast, Mer+ tumor cells continued to grow in all the 8 mice treated, indicating that Mer- tumor cells may be selectively inactivated by suitable therapeutic regimens with appropriate methylating drugs.

L4 ANSWER 8 OF 24 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1980:72508 CAPLUS
DOCUMENT NUMBER: 92:72508
TITLE: Kinetics of **adenovirus** DNA replication. I. Rate of **adenovirus** DNA replication
AUTHOR(S): Bodnar, John W.; Pearson, George D.
CORPORATE SOURCE: Dep. Biochem. Biophys., Oregon State Univ., Corvallis, OR, 97331, USA
SOURCE: Virology (1980), 100(1), 208-11
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The rate of **adenovirus** DNA replication in **HeLa S3** cells was constant throughout infection. The average rate of replication was 0.046 fractional lengths/min or 1600 nucleotides/min. The time required to synthesize an **adenovirus** DNA mol. was 21.7 min.

L4 ANSWER 9 OF 24 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1978:149950 CAPLUS
DOCUMENT NUMBER: 88:149950
TITLE: Involvement of microtubules in cytopathic effects of animal viruses: early proteins of **adenovirus** and herpesvirus inhibit formation of microtubular paracrystals in **HeLa-S3** cells
AUTHOR(S): Ebina, T.; Satake, M.; Ishida, N.
CORPORATE SOURCE: Dep. Bacteriol., Tohoku Univ. Sch. Med., Sendai, Japan
SOURCE: Journal of General Virology (1978), 38(3), 535-48
CODEN: JGVIAY; ISSN: 0022-1317
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In order to exam. the involvement of microtubles in the virus-induced cytopathic effect (c.p.e.), the effect of virus infection on the formation of microtubular paracrystals (PC) induced by 10 μg/mL of vinblastine sulfate in **HeLa-S3** cells was examined by phase-contrast microscopy. In poliovirus-infected cells, c.p.e. (cell rounding) and the inhibition of PC formation proceeded in parallel, starting 4 h post-infection. In Sendai virus-infected cells, however, PC formation was not inhibited even 24 h postinfection, when most infected cells clearly showed c.p.e. (syncytial formation). In **adenovirus**-infected cells, the inhibition of PC formation was observed 9 h before the appearance of c.p.e. Cytosine arabinoside (ara C) did not block the inhibition of PC formation in infected cells, but blocked the appearance of late c.p.e. (nuclear alteration). Cycloheximide blocked both the inhibition of PC formation and the induction of late c.p.e. These results suggest that an early protein synthesized de novo by **adenovirus** is required for direct or indirect inhibition of the microtubular PC formation. Furthermore, on UV inactivation of **adenovirus** both activities (induction of early c.p.e. shown by shrinkage of cytoplasm, and inhibition of PC formation) followed the same inactivation curve and were inactivated at a slower rate than viral infectivity and the activity leading to late c.p.e. The UV light sensitive target responsible for the induction of early c.p.e. and the inhibition of PC formation was .apprx.20% of that for infectivity and was in accord with the genome size of the early functioning virus genes. In herpes simplex virus (HSV)-infected cells, the inhibition of PC formation, the appearance of c.p.e. (cell rounding

and disappearance of nucleoli), and the synthesis of V antigen proceeded in parallel. These 3 functions of HSV were not blocked in infected cells even when the de novo synthesis of virus DNA was inhibited by ara C or phosphonoacetic acid (PAA), whereas these 3 functions were blocked by cycloheximide, suggesting that a protein coded by the input virus genome early after infection inhibits the microtubular PC formation and is responsible for c.p.e. From the UV inactivation curve of HSV, it was confirmed that only one-tenth of the virus genome was responsible for both activities.

L4 ANSWER 10 OF 24 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1978:133059 CAPLUS
DOCUMENT NUMBER: 88:133059
TITLE: Characterization of **adenovirus** RNA synthesized in the presence of an adenosine analog: failure of poly(A) addition
AUTHOR(S): Swart, C.; Hodge, L. D.
CORPORATE SOURCE: Dep. Hum. Genet., Yale Univ. Sch. Med., New Haven, CT, USA
SOURCE: Virology (1978), 84(2), 374-89
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Synthesis of **adenovirus**-specific RNA in the presence of toyocamycin, an adenosine analog, late in infection of **HeLa S3** cells has been investigated. The effect of this analog on nuclear metabolism has been examined because, under the appropriate conditions, there is an apparent accumulation of rapidly sedimenting nuclear viral RNA (HnRNA) and no new viral mRNA assocs. with polyribosomes. Under these conditions there was an .apprx.10% substitution by toyocamycin for adenosine in viral HnRNA. A similar amount was incorporated into virus-associated RNA(s) but there was little effect on the synthesis, the size, or the appearance in the cytoplasm of this species of viral RNA. In the presence of the analog no polyadenylate-rich segments could be detected in nuclear viral RNA. Two 5' termini containing the methylated components 7-methyl-GMP and 6-methyl-AMP were recovered and constituted proportionately the same amount in selected RNA sequences whether or not synthesis had occurred in the presence of the adenosine analog. Relative to the recovery of 5' termini, selectively extracted RNA synthesized in the presence of toyocamycin yielded nearly 2-fold less 6-methyl-AMP. Since rapid sedimentation of nuclear viral RNA implies incomplete processing of mols., these results suggest that the incorporation of toyocamycin interferes with RNA metabolism because of its prevention of polyadenylation and(or) reduction in methylation of internal adenosine residues. The data also imply a sequence of events in which the introduction of at least some 5' alterations and internal methylations can occur prior to and independent of polyadenylate addns.

L4 ANSWER 11 OF 24 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1977:28332 CAPLUS
DOCUMENT NUMBER: 86:28332
TITLE: Nuclear matrix of **HeLa S3** cells. Polypeptide composition during **adenovirus** infection and in phases of the cell cycle
AUTHOR(S): Hodge, L. D.; Mancini, P.; Davis, F. M.; Heywood, P.
CORPORATE SOURCE: Sch. Med., Yale Univ., New Haven, CT, USA
SOURCE: Journal of Cell Biology (1977), 72(1), 194-208
CODEN: JCLBA3; ISSN: 0021-9525
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A subnuclear fraction has been isolated from **HeLa S3** nuclei after treatment with high salt buffer, deoxyribonuclease, and dithiothreitol. Ultrastructural and biochem. analyses indicated that this structure consisted of nonmembranous and membranous elements. Its chemical composition was 87% protein, 12% phospholipid, 1% DNA, and 0.1% RNA by weight The protein constituents were resolved in Na dodecyl sulfate polyacrylamide slab gels into 30-35 distinguishable bands in the apparent mol. weight range of 14,000-200,000 with major peptides at 14,000-18,000 and 45,000-75,000. Anal. of newly synthesized polypeptides by cylindrical gel electrophoresis

revealed another cluster in the 90,000-130,000 mol. weight range. Infection with **adenovirus** resulted in an altered polypeptide profile. Addnl. polypeptides with apparent mol. wts. of 21,000, 23,000, and 92,000 became major components by 22 h after infection and some peptides in the 45,000-75,000 mol. weight range became less prominent. In synchronized cells the relative staining capacity of the 6 bands in the 45,000-75,000 mol. weight range changed during the cell cycle. Synthesis of at least some matrix polypeptides occurred in all phases of the cell cycle, although there was decreased synthesis in late S/G2. In the absence of protein synthesis after cell division, at least some polypeptides in the 45,000-75,000 mol. weight range survive nuclear dispersal and subsequent reformation during mitosis.

L4 ANSWER 12 OF 24 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1968:504862 CAPLUS
DOCUMENT NUMBER: 69:104862
TITLE: Studies on photosensitizing dyes. III. Effect of photosensitizing dyes on infected cells
AUTHOR(S): Ogasawara, Hisayasu
CORPORATE SOURCE: Med. Sch., Okayama Univ., Okayama, Japan
SOURCE: Kanko Shikiso (1968), No. 73, 17-20
CODEN: KASHAJ; ISSN: 0461-5956
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB The inhibitory effect of the photosensitizing dyes of cyanine and aminovinyl compds. on the growth of **HeLa-S3** cells, in tissue culture infected with **adenovirus** type 12 or with measles virus, was studied. The cyanine compds., such as 4,4-dimethyl-3,3'-di-n-heptyl-2,2'-thiazolocyanine nicotinate, 1,1-diethyl-11-[4-(1-ethyl quinoline)]-4,4'-dicarbocyanine di-L-aspartate, and 1,1'-diethyl-11-[4-(1-ethyl quinoline)]-4,4'-dicarbocyanine diglucuronate, 6-methyl-1-isopropyl-2-[2-(5-bromo-2-pyridylamino)vinyl]-pyridinium iodide, 1-ethyl-6-methyl-2-[2-(5-iodo pyrimidylamino)vinyl]-pyridinium iodide, 3,4-dimethyl-2-(2-pyrimidyl-aminovinyl) oxazolium iodide, and 3,4-dimethyl-2-(2-anilino vinyl) oxazolium iodide, showed a slight inhibitory effect on the proliferation of **HeLa-S3** cells infected with **adenovirus** type 12, while that of cells infected with measles virus was not affected by these photosensitizing dyes.

L4 ANSWER 13 OF 24 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2005:492837 BIOSIS
DOCUMENT NUMBER: PREV200510288621
TITLE: Association of the human papillomavirus type 16 E7 oncoprotein with the 600-kDa retinoblastoma protein-associated factor, p600.
AUTHOR(S): Huh, Kyung-Won; DeMasi, Joseph; Ogawa, Hiclesato; Nakatani, Yoshihiro; Howley, Peter M.; Munger, Karl [Reprint Author]
CORPORATE SOURCE: Brigham and Womens Hosp, Channing Lab, 75 Francis St, Boston, MA 02115 USA
kmunger@rics.bwh.harvard.edu
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (AUG 9 2005) Vol. 102, No. 32, pp. 11492-11497.
CODEN: PNASA6. ISSN: 0027-8424.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Nov 2005
Last Updated on STN: 16 Nov 2005
AB The human papillomavirus type 16 (HPV-16) E7 gene encodes a multifunctional oncoprotein that can subvert multiple cellular regulatory pathways. The best-known cellular targets of the HPV-16 E7 oncoprotein are the retinoblastoma tumor suppressor protein pRB and the related pocket proteins p107 and p130. However, there is ample evidence that E7 has additional cellular targets that contribute to its transforming potential. We isolated HPV-16 E7 associated cellular protein complexes by tandem affinity purification and mass spectrometry and identified the 600-kDa retinoblastoma protein associated factor, p600, as a cellular target of

E7. Association of E7 with p600 is independent of the pocket proteins and is mediated through the N terminal E7 domain, which is related to conserved region 1 of the **adenovirus** E1A protein and importantly contributes to cellular transformation independent of pRB binding. Depletion of p600 protein levels by RNA interference substantially decreased anchorage-independent growth in HPV-positive and -negative human cancer cells. Therefore, p600 is a cellular target of E7 that regulates cellular pathways that contribute to anchorage-independent growth and cellular transformation.

L4 ANSWER 14 OF 24 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1999:111560 BIOSIS
DOCUMENT NUMBER: PREV199900111560
TITLE: Expression of coxsackie-**adenovirus** receptor (CAR) and integrins in a cell line derived from anaplastic large cell lymphoma (ALCL).
AUTHOR(S): Turturro, F.; Link, C. J.
CORPORATE SOURCE: Human Gene Therapy Res. Inst. John Stoddard Cancer Cent., Iowa Methodist Med. Cent., Des Moines, IA, USA
SOURCE: Blood, (Nov. 15, 1998) Vol. 92, No. 10 SUPPL. 1 PART 1-2, pp. 381B. print.
Meeting Info.: 40th Annual Meeting of the American Society of Hematology. Miami Beach, Florida, USA. December 4-8, 1998. The American Society of Heamatology.
CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Mar 1999
Last Updated on STN: 12 Mar 1999

L4 ANSWER 15 OF 24 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1999:37471 BIOSIS
DOCUMENT NUMBER: PREV199900037471
TITLE: Inhibition of host RNA polymerse II-dependent transcription by vesicular stomatitis virus results from inactivation of TFIID.
AUTHOR(S): Yuan, Hang [Reprint author]; Yoza, Barbara K.; Lyles, Douglas S.
CORPORATE SOURCE: Dep. Microbiol. Immunol., Wake Forest Univ. Sch. Med., Winston-Salem, NC 27157, USA
SOURCE: Virology, (Nov. 25, 1998) Vol. 251, No. 2, pp. 383-392. print.
CODEN: VIRLAX. ISSN: 0042-6822.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 3 Feb 1999
Last Updated on STN: 3 Feb 1999

AB During infection with vesicular stomatitis virus (VSV), host-cell mRNA synthesis is inhibited due to shut off of host-cell transcription. The transcriptional activity of nuclear extracts prepared from VSV-infected cells was compared to the activity of nuclear extracts from uninfected cells. An exogenous DNA template was used which contained an **adenovirus** major late promoter (AdMLP) but lacked upstream activating sequences, so that only basal transcription activity was assayed in these experiments. AdMLP-initiated transcription was decreased by 75% in nuclear extracts from infected cells as early as 3 h p.i. and by >90% by 6 h p.i. Mixing nuclear extracts from uninfected and VSV-infected cells revealed that the inhibition was caused by lack of an active form of a host factor involved in basal transcription rather than by the presence of an excess of inhibitory factor. To determine which transcription factors were lacking from nuclear extracts of infected cells, host transcription initiation factors isolated from uninfected cells by ion-exchange chromatography were added separately to nuclear extracts inactivated by VSV infection. A phosphocellulose column fraction from uninfected cells eluted with 0.8 M KCl, which contained transcription factor IID (TFIID), overcame the inhibition. The corresponding fraction

from infected cells had no detectable activity in a TFIID-dependent in vitro transcription assay. TATA-binding protein (TBP) is the DNA-binding subunit of TFIID and has been shown previously to substitute for TFIID in basal transcription. Purified recombinant TBP also reconstituted the transcription activity of nuclear extracts from infected cells, supporting the idea that TFIID is the target of virus-induced inhibition. Western blot analysis showed that the level of TBP in nuclear extracts or in the 0.8 M KCl column fraction was not changed by VSV infection. These results indicated that VSV infection leads to an inhibition of host transcription by inactivation of TFIID rather than reduction in the level of TFIID.

L4 ANSWER 16 OF 24 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1997:45643 BIOSIS
DOCUMENT NUMBER: PREV199799344846
TITLE: Method for targeted gene transfer to liver using DNA-protein complexes.
AUTHOR(S): Findeis, Mark A. [Reprint author]; Wu, Catherine H.; Wu, George Y.
CORPORATE SOURCE: Pharm. Peptides Inc., Cambridge, MA, USA
SOURCE: Robbins, P. D. [Editor]. (1997) pp. 135-152. Methods in Molecular Medicine; Gene therapy protocols.
Publisher: Humana Press Inc., Suite 808, 999 Riverview Drive, Totowa, New Jersey 07512, USA.
ISBN: 0-89603-307-4 (paper), 0-89603-484-4 (cloth).
DOCUMENT TYPE: Book; (Book Chapter)
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Feb 1997
Last Updated on STN: 4 Feb 1997

L4 ANSWER 17 OF 24 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1996:462002 BIOSIS
DOCUMENT NUMBER: PREV199699184358
TITLE: Selection of the best target site for ribozyme-mediated cleavage within a fusion gene for **adenovirus** E1A-associated 300 kDa protein (p300) and luciferase.
AUTHOR(S): Kawasaki, Hiroaki; Ohkawa, Jun; Tanishige, Norie; Yoshinari, Koichi; Murata, Takehide; Yokoyama, Kazunari K.; Taira, Kazunari [Reprint author]
CORPORATE SOURCE: Inst. Applied Biochem., Univ. Tsukuba, Tennoudai 1-1-1, Tsukuba Science City 305, Japan
SOURCE: Nucleic Acids Research, (1996) Vol. 24, No. 15, pp. 3010-3016.
CODEN: NARHAD. ISSN: 0305-1048.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Oct 1996
Last Updated on STN: 11 Oct 1996

AB The cellular 300 kDa protein known as p300 is a target for the adenoviral E1A oncoprotein and it is thought to participate in prevention of the G-0/G-1 transition during the cell cycle, in activation of certain enhancers and in the stimulation of differentiation pathways. In order to determine the exact function of p300, as a first step we constructed a simple assay system for the selection of a potential target site of a hammerhead ribozyme in vivo. For the detection of ribozyme-mediated cleavage, we used a fusion gene (p300-luc) that consisted of the sequence encoding the N-terminal region of p300 and the gene for luciferase, as the reporter gene. We were also interested in the correlation of the GUX rule, for the triplet adjacent to the cleavage site, with ribozyme activity in vivo. Therefore, we selected five target sites that all included GUX. The rank order of activities in vitro indeed followed the GUX rule; with respect to the k-cat, a C residue as the third base (X) was the best, next came an A residue and a U residue was the worst (GUC gt GUA gt GUU). However, in vivo the tRNA-Val promoter-driven ribozyme, targeted to a GUA located upstream of the initiation codon, had the highest inhibitory effect (96%) in **HeLa S3** cells when the molar ratio of the DNA template for the target p300 RNA to that for the ribozyme was 1:4. Since the rank order of activities in vivo did not

conform to the GUX rule, it is unlikely that the rate limiting step for cleavage of the p300-luc mRNA was the chemical step. This kind of ribozyme expression system should be extremely useful for elucidation of the function of p300 in vivo.

L4 ANSWER 18 OF 24 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1995:439684 BIOSIS
DOCUMENT NUMBER: PREV199598453984
TITLE: Poliovirus Protease 3C Mediates Cleavage of Microtubule-Associated Protein 4.
AUTHOR(S): Joachims, Michelle; Harris, Kevin S.; Etchison, Diane [Reprint author]
CORPORATE SOURCE: Dep. Mol. Biol., Univ. California Irvine, Irvine, CA 92717, USA
SOURCE: Virology, (1995) Vol. 211, No. 2, pp. 451-461. CODEN: VIRLAX. ISSN: 0042-6822.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Oct 1995
Last Updated on STN: 1 Nov 1995
AB Poliovirus infection results in a number of host cell changes, including specific alterations in cellular proteins. This study further characterizes the cleavage of a cytoskeletal protein, microtubule-associated protein 4 (MAP-4) and investigates the identity of the viral protease which mediates its cleavage. MAP-4 cleavage by poliovirus was previously identified using a monoclonal antibody (M. Joachims and D. Etchison, 1992, J. Virol. 66, 5997-5804). In this study, MAP-4 cleavage was found to occur in cells infected by only some picornaviruses, poliovirus and human rhinovirus 14. Infection by other types of viruses, vesicular stomatitis virus and **adenovirus**, or by other types of picornaviruses, encephalomyocarditis virus, did not result in MAP-4 cleavage. To determine the viral mediator of MAP-4 cleavage, the effects of purified poliovirus proteases on MAP-4 integrity were examined by immunoblot. When MAP-4 substrates were incubated with concentrations of poliovirus 2A that were more than sufficient to induce p220 cleavage, there was no effect on MAP-4. However, when MAP-4 substrates were incubated with purified 3C protease (3C-pro), cleavage products were detected that were identical in size to those generated in vivo in poliovirus-infected cells; the use of a mutant 3C protease did not result in MAP-4 cleavage. Cleavage of MAP-4 was also demonstrated with purified 3CD-pro, and the in vitro cleavage kinetics were examined. Indirect immunofluorescence revealed that MAP-4 cleavage also correlated with a marked "collapse" of microtubules during late infection, indicating a possible relationship between 3C-pro-mediated MAP-4 cleavage and changes in the microtubule system of infected cells.

L4 ANSWER 19 OF 24 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1994:443911 BIOSIS
DOCUMENT NUMBER: PREV199497456911
TITLE: Vitronectin receptor antibodies inhibit infection of HeLa and A549 cells by **adenovirus** type 12 but not by **adenovirus** type 2.
AUTHOR(S): Bai, Mei; Campisi, Lauren; Freimuth, Paul [Reprint author]
CORPORATE SOURCE: Biol. Dep., Brookhaven National Lab., Upton, New York 11973, USA
SOURCE: Journal of Virology, (1994) Vol. 68, No. 9, pp. 5925-5932. CODEN: JOVIAM. ISSN: 0022-538X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Oct 1994
Last Updated on STN: 12 Oct 1994
AB The penton base gene from **adenovirus** type 12 (Ad12) was sequenced and encodes a 497-residue polypeptide, 74 residues shorter than the penton base from Ad2. The Ad2 and Ad12 proteins are highly conserved at the amino- and carboxy-terminal ends but diverge radically in the central region, where 63 residues are missing from the Ad12 sequence. Conserved within this variable region is the sequence Arg-Gly-Asp (RGD),

which, in the Ad2 penton base, binds to integrins in the target cell membrane, enhancing the rate or the efficiency of infection. The Ad12 penton base was expressed in Escherichia coli, and the purified refolded protein assembled in vitro with Ad2 fibers. In contrast to the Ad2 penton base, the Ad12 protein failed to cause the rounding of adherent cells or to promote attachment of **HeLa S3** suspension cells; however, A549 cells did attach to surfaces coated with either protein and pretreatment of the cells with an integrin alpha-v-beta-5 monoclonal antibody reduced attachment to background levels. Treatment of HeLa and A549 cells with integrin alpha-v-beta-3 or alpha-v-beta-5 monoclonal antibodies or with an RGD-containing fragment of the Ad2 penton base protein inhibited infection by Ad12 but had no effect on and in some cases enhanced infection by Ad2. Purified Ad2 fiber protein reduced the binding of radiolabeled Ad2 and Ad12 virions to HeLa and A549 cells nearly to background levels, but the concentrations of fiber that strongly inhibited infection by Ad2 only weakly inhibited Ad12 infection. These data suggest that alpha-v-containing integrins alone may be sufficient to support infection by Ad12 and that this pathway is not efficiently used by Ad2.

L4 ANSWER 20 OF 24 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1985:371506 BIOSIS
DOCUMENT NUMBER: PREV198580041498; BA80:41498
TITLE: ANALYSIS OF N METHYL-N'-NITRO-N-NITROSOGUANIDINE-INDUCED DNA DAMAGE IN TUMOR CELL STRAINS FROM JAPANESE PATIENTS AND DEMONSTRATION OF N METHYL-N'-NITRO-N-NITROSOGUANIDINE HYPERSENSITIVITY OF MER-NEGATIVE XENOGRAFTS IN ATHYMIC NUDE MICE.
AUTHOR(S): WATATANI M [Reprint author]; IKENAGA M; HATANAKA T; KINUTA M; TAKAI S-I; MORI T; KONDO S
CORPORATE SOURCE: DEP FUNDAMENTAL RADIOL, FAC MED, OSAKA UNIV, NAKANOSHIMA 4-3-57, KITA-KU, OSAKA 530, JPN
SOURCE: Carcinogenesis (Oxford), (1985) Vol. 6, No. 4, pp. 549-554. CODEN: CRNGDP. ISSN: 0143-3334.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB Among 15 human tumor cell strains from Japanese patients, 1 strain derived from a patient with thyroid cancer showed inability to support the growth of **adenovirus** 5 treated with N-methyl-N'-nitro-N-nitrosoguanidine (MNNG). When plated on this Mer- strain, **adenovirus** 5 showed 3-4 times higher sensitivity to MNNG-induced killing than when plated on any of the other 14 Mer+ tumor cell strains. Biochemical analysis showed that the Mer- strain was defective in demethylation repair of O6-methylguanine produced by MNNG treatment. The sensitivities of 12 of the 15 human tumor strains, including the Mer- strain, to MNNG were compared by measuring their colony-forming abilities. All the strains tested showed the Rem- phenotype (having higher sensitivity to MNNG-produced cell killing than normal fibroblasts). The differential killing effects of MNNG on Mer- and Mer+ tumor cells under in vivo conditions were tested using the Mer+ **HeLa S3** strain and its Mer- variant. Mer+ cells and Mer- cells were implanted subcutaneously into the left and right flanks, respectively, of 10 nude mice and the next day, MNNG solution (0.25 ml at 1 mg/ml) was injected into the implantation sites of 8 mice. Mer- tumor cells in 6 of 8 treated mice showed no growth and those in the other 2 mice did grow, but regressed after .apprx.. 3 wk. In contrast, Mer+ tumor cells continued to grow in all the 8 mice treated, indicating that Mer- tumor cells may be selectively inactivated by suitable therapeutic regimens with appropriate methylating drugs.

L4 ANSWER 21 OF 24 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1978:187464 BIOSIS
DOCUMENT NUMBER: PREV197865074464; BA65:74464
TITLE: CHARACTERIZATION OF **ADENOVIRUS** RNA SYNTHESIZED IN THE PRESENCE OF AN ADENOSINE ANALOG FAILURE OF POLY ADENYLATE ADDITION.
AUTHOR(S): SWART C [Reprint author]; HODGE L D

CORPORATE SOURCE: DEP HUM GENET, YALE UNIV SCH MED, NEW HAVEN, CONN 06510, USA
SOURCE: Virology, (1978) Vol. 84, No. 2, pp. 374-389.
CODEN: VIRLAX. ISSN: 0042-6822.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB Synthesis of **adenovirus**-specific RNA in the presence of toyocamycin, an adenosine analog, late in infection of [human cervical carcinoma] **HeLa S3** cells was investigated. The effect of this analog on nuclear metabolism was examined because, under the appropriate conditions, there is an apparent accumulation of rapidly sedimenting nuclear viral RNA (HnRNA) and no new viral mRNA associates with polyribosomes. Under these conditions there was an approximate 10% substitution by toyocamycin for adenosine in viral HnRNA. A similar amount was incorporated into virus-associated RNA but there was little effect on the synthesis, the size or the appearance in the cytoplasm of this species of viral RNA. In the presence of the analog no poly(A)-rich segments could be detected in nuclear viral RNA. Two 5' termini containing the methylated components 7mG and m6Am were recovered and constituted proportionately the same amount in selected RNA sequences whether or not synthesis had occurred in the presence of the adenosine analog. Relative to the recovery of 5' termini, selectively extracted RNA synthesized in the presence of toyocamycin yielded nearly 2-fold less m6Ap. Since rapid sedimentation of nuclear viral RNA implies incomplete processing of molecules, these results suggest that the incorporation of toyocamycin interferes with RNA metabolism because of its prevention of polyadenylation and/or reduction in methylation of internal adenosine residues. There is apparently a sequence of events in which the introduction of at least some 5' alterations and internal methylations can occur prior to and independent of poly(A) additions.

L4 ANSWER 22 OF 24 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1978:175072 BIOSIS
DOCUMENT NUMBER: PREV197865062072; BA65:62072
TITLE: INVOLVEMENT OF MICRO TUBULES IN CYTOPATHIC EFFECTS OF ANIMAL VIRUSES EARLY PROTEINS OF **ADENOVIRUS** AND HERPESVIRUS INHIBIT FORMATION OF MICRO TUBULAR PARA CRYSTALS IN HELA-S-3 CELLS.
AUTHOR(S): EBINA T [Reprint author]; SATAKE M; ISHIDA N
CORPORATE SOURCE: DEP BACTERIOL, TOHOKU UNIV SCH MED, 2-1 SEIRYO-MACHI, SENDAI, MIYAGI 980, JPN
SOURCE: Journal of General Virology, (1978) Vol. 38, No. 3, pp. 535-548.
CODEN: JGVIAY. ISSN: 0022-1317.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB To examine the involvement of microtubules in the virus-induced cytopathic effect (c.p.e.), the effect of virus infection on the formation of microtubular paracrystals (PC) induced by 10 μg/ml of vinblastine sulfate in [human cervical carcinoma] **HeLa-S3** cells was examined by phase-contrast microscopy. In poliovirus-infected cells, c.p.e. (cell rounding) and the inhibition of PC formation proceeded in parallel, starting 4 h post-infection. In Sendai virus-infected cells, PC formation was not inhibited even 24 h post-infection when most infected cells clearly showed c.p.e. (syncytial formation). In **adenovirus** -infected cells, the inhibition of PC formation was observed 9 h before the appearance of c.p.e. Cytosine arabinoside (ara C) did not block the inhibition of PC formation in infected cells, but blocked the appearance of late c.p.e. (nuclear alteration). Cycloheximide blocked both the inhibition of PC formation and the induction of late c.p.e. An early protein synthesized de novo by **adenovirus** is required for direct or indirect inhibition of the microtubular PC formation. On UV inactivation of **adenovirus** both activities (induction of early c.p.e. shown by shrinkage of cytoplasm, and inhibition of PC formation) followed the same inactivation curve and were inactivated at a slower rate than viral infectivity and the activity leading to late c.p.e. The UV

light sensitive target responsible for the induction of early c.p.e. and the inhibition of PC formation is about 20% of that for infectivity and is in accord with the genome size of the early functioning virus genes. In herpes simplex virus (HSV)-infected cells, the inhibition of PC formation, the appearance of c.p.e. (cell rounding and disappearance of nucleoli) and the synthesis of V antigen proceeded in parallel. These 3 functions of HSV were not blocked in infected cells even when the de novo synthesis of virus DNA was inhibited by ara C or phosphonoacetic acid (PAA), but these 3 functions were blocked by cycloheximide, suggesting that a protein coded by the input virus genome early after infection inhibits the microtubular PC formation and is responsible for c.p.e. The UV inactivation curve of HSV, showed that only 1/10 of virus genome was responsible for both activities (induction of c.p.e. and inhibition of PC formation).

L4 ANSWER 23 OF 24 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1978:175035 BIOSIS
DOCUMENT NUMBER: PREV197865062035; BA65:62035
TITLE: FREEZE ETCHING OBSERVATION ON THE NUCLEUS OF HELA-S-3 CELL INFECTED WITH **ADENOVIRUS** TYPE 12.
AUTHOR(S): FUJIO K [Reprint author]; ICHIKAWA H; KUMON H; UNO F; TAWARA J
CORPORATE SOURCE: DEP VIROL, OKAYAMA UNIV MED SCH, OKAYAMA 700, JPN
SOURCE: Journal of Electron Microscopy, (1976) Vol. 25, No. 4, pp. 297-298.
CODEN: JELJA7. ISSN: 0022-0744.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB Human cervical carcinoma **HeLa-S3** cells were inoculated with **adenovirus** type 12. An electron micrograph of crystal arrangement in the nucleus at low magnification showed about 500 **adenovirus** particles in a crystal. The virions are of hexagonal shape, with an average diameter of 80 nm. The capsomeres are observed as small granules, but it is difficult to distinguish penton from hexon. Freeze-etching techniques are suitable for observation of the process of virus maturation in vells.

L4 ANSWER 24 OF 24 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1977:144575 BIOSIS
DOCUMENT NUMBER: PREV197763039439; BA63:39439
TITLE: NUCLEAR MATRIX OF HELA-S-3 CELLS POLY PEPTIDE COMPOSITION DURING **ADENOVIRUS** INFECTION AND IN PHASES OF THE CELL CYCLE.
AUTHOR(S): HODGE L D; MANCINI P; DAVIS F M; HEYWOOD P
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AB A subnuclear fraction was isolated from **HeLa S3** nuclei after treatment with high salt buffer, DNase and dithiothreitol. This fraction retains the approximate size and shape of nuclei and resembles the nuclear matrix recently isolated from rat liver nuclei. Ultrastructural and biochemical analyses indicate that this structure consists of nonmembranous and membranous elements. Its chemical composition is 87% protein, 12% phospholipid, 1% DNA and 0.1% RNA by weight. The protein constituents are resolved in SDS[sodium dodecyl sulfate]-polyacrylamide slab gels into 30-35 distinguishable bands in the apparent MW range of 14,000-200,000, with major peptides at 14,000-18,000 and 45,000-75,000. Analysis of newly synthesized polypeptides by cylindrical gel electrophoresis reveals another cluster in the 90,000-130,000 MW range. Infection with **adenovirus** [type 2] results in an altered polypeptide profile. Additional polypeptides with apparent MW of 21,000, 23,000 and 92,000 become major components by 22 h after infection. Concomitantly, some peptides in the 45,000-75,000 MW range become less prominent. In synchronized cells the relative staining

capacity of the 6 bands in the 45,000-75,000 MW range changes during the cell cycle. Synthesis of at least some matrix polypeptides occurs in all phases of the cell cycle, although there is decreased synthesis in late S/G2. In the absence of protein synthesis after cell division, at least some polypeptides in the 45,000-75,000 MW range survive nuclear dispersal and subsequent reformation during mitosis. The possible significance of this subnuclear structure with regard to structure-function relationships within the nucleus during virus replication and during the life cycle of the cell is discussed.